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Pregnancy outcomes of women presenting with stage 1 hypertension during the first prenatal clinic visit before 20 gestational weeks

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ABSTRACT

Objective: To determine the pregnancy outcomes of women who had 2017 American College of Cardiologists stage 1 hypertension during the first prenatal clinic visit before 20 gestational weeks in a tertiary hospital in South Africa.

Study design: A retrospective cohort study involving the review of medical records of 127 participants with stage 1 hypertension and 128 control with blood pressure (BP) less than stage 1 hypertension before 20 weeks' gestation.

Main outcome measures: The primary outcome measure was progression to stage 2 hypertension (BP $\geq$ 140/90 mmHg). Secondary outcome measures were a combination of maternal variables (postpartum BP $\geq$ 140/90 mmHg, use of antihypertensives within 24 h postpartum, pulmonary oedema, and maternal death within 24 h postpartum) and perinatal variables (fetal growth restriction, gestational age at delivery, fetal compromise, abruptio placenta, birth weight, Apgar score in 1 and 5 min).

Results: The study and control arms were similar in age, parity, and comorbidities ($p > 0.05$). The following maternal outcomes were worse ($p < 0.001$) in the study compared to control arm: progression to stage 2 hypertension (46 % vs 1.6 %), postpartum systolic BP $\geq$ 140 mmHg (33.9 % vs 1.6 %), postpartum diastolic BP $\geq$ 90 mmHg (22.1 % vs 1.6 %) and use of antihypertensives within 24 h postpartum (27.6 % vs 0.8 %). Other outcome measures did not differ between the two groups (p > 0.05).

Conclusions: Stage 1 hypertension occurring before 20 weeks' gestation increases the risk of progression to stage 2 hypertension in pregnancy and the use of antihypertensive drug therapy within 24 h postpartum.

1. Introduction

Hypertensive disorders of pregnancy (HDP) affect 5–10 % of pregnancies worldwide [1], and account for major adverse outcomes, which include 14 % of global maternal mortality [2]. Furthermore, preeclampsia alone causes over 70 000 maternal deaths and 500 000 fetal and neonatal deaths globally [3]. To improve risk stratification and treatment, and reduce cardiovascular disease events, in 2017, the American College of Cardiology (ACC) and American Heart Association (AHA) re-classified levels of BP in adults. This resulted in a new definition of hypertension with systolic BP levels 130–139 mmHg and or diastolic BP 80–89 mmHg categorized as stage 1 hypertension [4].

Stage 1 hypertension has been associated with an increase in the risk of cardiac event [5]. However, the new definition of hypertension (2017 ACC/AHA stage 1 hypertension) has not been accepted by many organizations including the International Society for Study of Hypertension in Pregnancy (ISSHP), which currently recognizes only sustained systolic BP $\geq$ 140 mmHg and or diastolic BP $\geq$ 90 mmHg as hypertension [3,6]. Nevertheless, the ISSHP recommended in 2021 that maternal BP level six months after childbirth should be < 120/80 mmHg [3]. This suggests that increasing BP is a risk for adverse events, which include cardiac events. Similarly, the 2019 South African National guidelines on HDP recognises prehypertension, also called stage 1b hypertension (systolic BP 135–139 mmHg and or diastolic BP 85–89 mmHg) as a risk factor for HDP and recommended that women presenting with this level of BP should be reviewed in 3 – 7 days to exclude progression to 2017 ACC/AHA stage 2 hypertension [7].

Notably, the findings of the Community-Level Interventions for Preeclampsia (CLIP) cluster randomised trial (n = 21 069 women) did not associate stage 1 hypertension with adverse pregnancy outcomes

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[8]. In contrast, a large retrospective cohort study (n = 47 874 women) reported that 40.1 % of women who had stage 1 hypertension at less than 20 weeks' gestation developed HDP during their pregnancies [9]. In another study, previously normotensive pregnant women diagnosed with new-onset ACC/AHA stage 1 hypertension after 20 gestational weeks were found to have an increased risk of developing HDP (adjusted RR 2.41, 95 % CI: 2.02 – 2.85) and also three times more likely to suffer from preeclampsia with severe features [10]. In SA, 12/44 (25.5 %) of teenagers that died following eclampsia during the 2014 – 2016 triennium were reported to have had ACC/AHA stage 1 hypertension [11]. This implies that the occurrence of stage 1 hypertension in the first prenatal clinic visit, which serves as a risk profiling encounter, is crucial and requires further investigation. Notably, 20 weeks' gestation is the threshold for categorizing HDP into new-onset and pre-existing types. To date, the pregnancy outcomes of 2017 ACC/AHA stage 1 hypertension have not been completely studied particularly in settings such as South Africa (SA). This is particularly important given the high burden of HDP in South Africa [12,13]. In this study, we aimed to determine the pregnancy outcomes of women who delivered their babies at a tertiary hospital in SA in 2019 and had ACC/AHA stage 1 hypertension during the first prenatal clinic visit before 20 weeks' gestation.

2. Methods

2.1. Ethical approval

The ethical approval to conduct the study was granted by the Human Research Ethics Committee (HREC) of University of the Witwatersrand (approval reference M210363).

2.2. Study design

Retrospective cohort study.

2.3. Study setting and duration

This study was conducted in Klerksdorp-Tshepong Hospital Complex, a tertiary health care facility in North West Province, SA. The data of women that gave birth at the hospital in 2019 was collected in 2021 and used for the study. Data from 2020 was not collected to prevent the influence that COVID-19 might have had on the study findings. The hospital manages both low- and high-risk pregnant women, and a total of 5851 deliveries were conducted in 2019.

2.4. Study population and sample

The study population comprised pregnant women that had 2017 ACC/AHA stage 1 hypertension (systolic BP 130 – 139 and or diastolic BP 80 – 89 mmHg) and those that had BP less than stage 1 hypertension (systolic BP < 130 and diastolic BP < 80 mmHg) during the first prenatal clinic visit before 20 weeks' gestation.

The sample was 255 participants divided into control and study arms. Those who had 2017 ACC/AHA stage 1 hypertension during the first prenatal clinic visit (known not to be chronic hypertensive) constituted the study arm (n = 127). The control arm (n = 128) were normotensive women with BP less than the threshold for stage 1 hypertension during the first prenatal clinic visit (Fig. 1).

The inclusion criteria were commencement of prenatal care before 20 weeks' gestation, having stage 1 hypertension in the first prenatal clinic visit, and giving birth in the study setting. We excluded women who had ACC/AHA stage 2 hypertension (chronic hypertension). Pregnant women who booked for prenatal care after 20 weeks' gestation were also excluded because they could have had BP more than stage 1 hypertension (such as transient gestation hypertension).

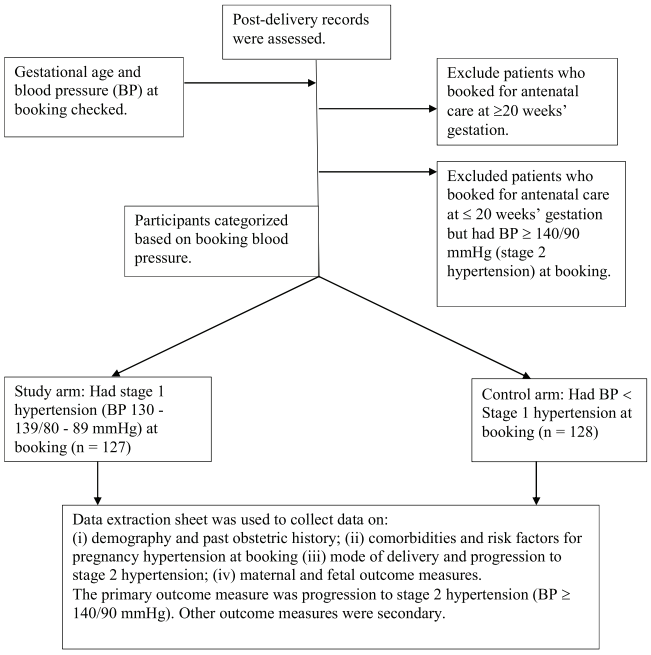

Fig. 1. Flow diagram of study participants and data collection.

2.5. Sampling method and data collection process

The birth register at the study setting was used to trace and retrieve the hospital medical records of women who gave birth in the study setting in 2019. Each medical record was reviewed to select those who met the inclusion criteria. Participants' medical records were selected using a convenient sampling method until the required sample was attained. A data collection sheet was used to collect the following information from the medical records: (i) demography and past obstetric history; (ii) comorbidities and risk factors for pregnancy hypertension at booking (iii) mode of delivery and progression to stage 2 hypertension; (iv) outcome measures.

2.6. Outcome measures

The primary outcome measure was progression to stage 2 hypertension (BP ≥ 140/90 mmHg). Secondary outcome measures included a combination of maternal variables (postpartum BP ≥ 140/90 mmHg, use of antihypertensives within 24 h postpartum, pulmonary oedema, and maternal death within 24 h postpartum); and perinatal variables (fetal growth restriction, gestational age at delivery, fetal compromise, abruptio placenta, birth weight, Apgar score in 1 and 5 min). Fig. 1 summarizes the data collection processes.

2.7. Sample size

The sample size was calculated based on the primary outcome measure, progression to HDP. In a previous large study (n = 47 874 pregnant women), 7.5 % of the participants who had BPs that were less than the threshold for stage 1 hypertension and 40.1 % of those with BP levels of stage 1 hypertension developed HDP (stage 2 hypertension) during pregnancy after commencing prenatal care before 20 weeks' gestation [9]. To calculate the sample size, we used the unmatched cohort and cross-sectional studies calculator in Epi Info version 7 (CDC, USA), and the input parameters were: power 99 %, the ratio of unexposed to exposed of 1, percentage of outcome in unexposed 7.5 %, and percentage of outcome in exposed 40.1 %. This generated a sample of 63 for each arm of the study. After considering the cost of the study, the possibility of missing data, and details about the sample size used in

other similar studies, we increased (doubled) the sample size to at least 126 in each arm. Therefore, in this study, the control and the study arms were 128 and 127 participants respectively.

2.8. Statistical analysis

Data was captured in Microsoft Excel, imported, and analysed using SPSS version 27.0 (IBM, Armonk, NY, USA). Descriptive analysis including an assessment of the normality of continuous variables was performed. The difference in each categorical variable between the study and control arms was analysed using the Chi-square test. The differences in the continuous variables between the two groups were determined using a one-way analysis of variance (one-way ANOVA). Of note, one-way ANOVA functions as a two-sample *t*-test when used to compare two groups [14]. The significance level was set at a p-value less than or equal to 0.05.

3. Results

Women in the study and control arms commenced antenatal care at less than 20 weeks' gestation. The demography and past obstetric history are presented in Table 1, with statistically significant differences ($p < 0.001$) observed between the two arms of the study in terms of maternal body weight and body mass index (BMI). There was no difference between the two groups with regards to the presence of comorbidities (including body mass index ≥ 25 kg/m^2), $p > 0.05$ (Table 2). Women in the study arm (stage 1 hypertension) had higher mean systolic and diastolic BPs at booking than those in the control arm, $p < 0.001$. Table 3 shows the mode of delivery and progression to stage 2 hypertension. The mean of the highest BPs and progression to stage 2 hypertension (HDP) were highest among women with stage 1 hypertension, $p < 0.001$. Table 4 depicts the maternal and fetal outcome measures. The primary outcome measure (progression to HDP) was statistically different ($p < 0.001$). In the study arm, 60/127 (47.2 %) of the women developed HDP compared to 2/128(1.6 %) in the control arm. Of these women that had HDP, 17 in the study arm and one in the control arm developed preeclampsia. Of the women who developed preeclampsia, 12 (9.5 %) in the study and none in the control arm had fetal growth restriction. There were no cases of preeclampsia that were diagnosed postpartum.

The secondary outcome measures that were statistically significant ($p < 0.001$) and more prevalent in the study than the control arm include the use of antihypertensive drugs within 24 h postpartum, systolic BP ≥ 140 mmHg within 24 h postpartum and diastolic BP ≥ 90 mmHg postpartum within 24 h postpartum.

4. Discussion

4.1. Main findings

Progression to stage 2 hypertension (HDP) was more likely among women that had stage 1 hypertension before 20 weeks' gestation (study arm) than in those that had BP less than stage 1 hypertension before 20 gestational weeks (control arm). The type of HDP developed by the women in the study arm, from most to least common, were gestational hypertension, preeclampsia, and transient gestational hypertension. Development of postpartum hypertension and use of antihypertensive drugs within 24 h postpartum were more common among women in the study arm compared to those in the control arm. However, there was no difference in fetal outcome measures between the two arms.

4.2. Strengths and limitations

There is limited data on pregnancy outcomes of stage 1 hypertension in low- and middle-income countries (LMIC). Our study is, therefore among the few from LMIC and contributes valuable data. However, the

Table 1
Demography and past obstetric history.

Parameter	Mean ± SD, or frequency n (%)		p-value
	Stage 1 ACC/AHA hypertension (study arm), n = 127	Blood pressure less than stage 1 hypertension (control arm), n = 128	
Age (years)	27.4 ± 1.0	27.6 ± 1.1	0.697
Number of all previous pregnancies	1.3 ± 0.2	1.53 ± 0.2	0.264
Parity			0.805
Primigravida	39 (30.7)	36(28.1)	
Multigravida	86 (67.7)	85(66.4)	
Missing data	2 (1.6)	7(5.5)	
Weight (kg) at first antenatal clinic visit	75.4 ± 3.4	63.1 ± 2.6	<0.001*
Body mass index (kg/m^2) at booking visit	29.9 ± 7.2	26.0 ± 5.6	<0.001*
Gestational age (weeks) at delivery	38.0 ± 0.5	38.2 ± 0.4	0.396
Occupation			0.899
Employed	34(26.8)	32 (25)	
Unemployed	85 (66.9)	83 (64.8)	
Missing data	8(6.3)	13(10.2)	
Marital status			0.448
Single	102 (80.3)	92 (71.8)	
Married	19 (15.0)	19 (14.8)	
Divorced	0(0.0)	1 (0.8)	
Not married but co-habiting with partner	2 (1.6)	5 (3.9)	
Missing data	4(3.2)	11(8.6)	
Residential address			0.663
Same town as the hospital	77(60.6)	77(60.2)	
Same health district as the hospital	39(30.7)	33(25.8)	
Outside the health district of the hospital	4(3.2)	7(5.5)	
Outside the state/ province as the hospital	2(1.6)	1(0.8)	
Missing data	5(3.9)	10(7.8)	
Past maternal complication			0.360
Preterm delivery (<37 weeks' gestation)	5(3.9)	7(5.5)	
Placental abruptio	0(0.0)	1(0.8)	
Others	38(29.9)	31(24.2)	
No previous maternal complication	84(66.4)	89(69.5)	
Past fetal complication			0.645
Early neonatal death	2(1.6)	1(0.8)	
Late neonatal death	3(2.4)	6(4.7)	
Admission to neonatal ICU	0(0)	0(0)	
Congenital anomaly	0(0)	1(0.8)	
Others	0(0)	1(0.8)	
No previous fetal complication	122(96.1)	119(93.0)	

Missing data were excluded from *p*-value calculation. Early neonatal death is death within the first seven days of life; Late neonatal death is death in the first 28 days of life.

Table 2
Comorbidities and risk factors for pregnancy hypertension at booking.

Parameter	Mean ± SD, or frequency n (%)		p-value
	Stage 1 ACC/AHA hypertension (study arm) n = 127	Blood pressure less than stage 1 hypertension (control arm), n = 128	
Haemoglobin	13.0 ± 1.4	14.7 ± 4.18	0.443
Presence of proteinuria at booking			0.391
Yes	1(0.8)	0(0)	
No	111(87.4)	105(82.0)	
Missing data	15(11.8)	23(18.0)	
Index pregnancy is multiple gestation			0.080
Yes	3(2.4)	0(0)	
No	124(96.9)	128(100)	
Other comorbidity	68(53.5)	75(58.6)	0.189
HIV	37(29.1)	41(32.0)	
Previous miscarriage	4(3.2)	16(12.5)	
High body mass index (≥25 kg/m^2)	10(7.9)	1(0.8)	
Previous caesarean delivery	5(3.9)	9(7.0)	
Anaemia	3(2.4)	4(3.1)	
Previous preterm birth	2(1.6)	1(0.8)	
Previous pulmonary tuberculosis	1(0.8)	1(0.8)	
Diabetes	0(0)	1(0.8)	
Epilepsy	1(0.8)	0	
Previous eclampsia	1(0.8)	0	
Previous intrauterine fetal demise	1(0.8)	0	
Previous LLETZ for CIN II	1(0.8)	0	
Previous myocardial infarction	1(0.8)	0	
Previous preeclampsia	1(0.8)	0	
Use of alcohol			0.579
Yes	12(9.5)	14(10.9)	
No	108(85.0)	100(78.1)	
Missing data	7(5.5)	14(10.9)	
Use of illicit substances			0.338
Yes	3(2.4)	1(0.8)	
No	117(92.1)	113(88.3)	
Missing data	7(5.5 %)	14(10.9)	
Family history of hypertension			0.813
Yes	6(4.7)	5(2.2)	
No	113(88.9)	109(46.8)	
Missing data	19(15.0)	14(10.9)	
Previous pregnancy hypertension			0.109
Yes	5(3.9)	1(0.4)	
No	114(89.8)	113(48.5)	
Missing data	8(6.3)	14(10.9)	
Calcium therapy to prevent preeclampsia			0.320
Yes	119(93.7)	118(92.2)	
No	3(2.4)	1(0.8)	
Missing data	5(3.9)	9(7.0)	
Aspirin therapy to prevent preeclampsia			0.630
Yes	2(1.6)	3(2.3)	
No	120(94.5)	116(90.6)	
Missing data	5(3.9)	9(7.0)	
Cigarette smoking during pregnancy			0.200
Yes	13(10.2)	7(5.5)	
No	106(83.5)	106(82.8)	
Missing data	8(6.3)	15(11.7)	
Blood pressure and pulse at booking			
Booking systolic blood pressure (mmHg)	124.5 ± 1.7	112.1 ± 1.5	<0.001
Booking diastolic blood pressure (mmHg)	72.6 ± 1.8	65.0 ± 1.5	<0.001
Pulse at booking (beats/minute)	87.5 ± 2.0	85.7 ± 2.0	0.219

Missing data were excluded from the calculation of p-value. Abbreviations: CIN, Cervical intraepithelial neoplasia; LLETZ, Large loop excision of transformation zone.

Table 3
Mode of delivery and progression to stage 2 hypertension.

Variable	Mean ± SD, or frequency n (%)		p-value
	Stage 1 ACC/AHA hypertension (study arm) n = 127	Blood pressure less than stage 1 hypertension (control arm), n = 128	
Highest blood pressure (BP) and pulse during pregnancy			
Highest systolic BP (mmHg)	136.0 ± 1.6	117.6 ± 1.7	<0.001
Highest diastolic BP (mmHg)	80.5 ± 1.9	67.9 ± 1.3	<0.001
Mode of delivery and indication for caesarean delivery			0.311
Elective caesarean delivery	21(16.5)	18(14.1)	
Emergency caesarean delivery	36(28.3)	24(18.8)	
Spontaneous vaginal delivery	65(51.2)	81(63.3)	
Vaginal delivery following induction of labour	2(1.6)	3(2.3)	
Missing data	3(2.4)	2(1.6)	
Main indications for caesarean delivery			0.006
Fetal compromise	28 (21.9)	14 (11.0)	
Previous caesarean delivery	19 (14.8)	17(13.4)	
Others	10(7.9)	11(8.6)	
Progression to stage 2 hypertension			<0.001
Yes	60(47.2)	2(1.6)	
No progression	61(48.0)	112(87.5)	
Missing data	6(4.7)	14(10.9)	
Gestational age at diagnosis of hypertensive disorder of pregnancy	33.3 ± 2.2	38 ± 0	0.322

Table 4
Maternal and fetal outcome measures.

Variable	Mean ± SD, or frequency (%)		p-value
	Stage 1 ACC/AHA hypertension (study arm) n = 127	Blood pressure less than stage 1 hypertension (control arm), n = 128	
Maternal outcome measure			
Progression to hypertensive disorders of pregnancy			<0.001
Yes	60(47.2)	2(1.6)	
Preeclampsia	17(13.4)	1(0.8)	
Gestational hypertension	37(29.1)	1(0.8)	
Transient gestational hypertension	6(4.7)	0	
No	61(48.0)	112(87.5)	
Missing data	6(4.7)	14(10.9)	
Use of antihypertensive drug within 24 h postpartum			<0.001
Yes	35(27.6)	1(0.8)	
No	89(70.1)	117(91.4)	
Missing data	3(2.4)	10(7.8)	
SBP ≥ 140 mmHg postpartum (within 24 h)			<0.001
Yes	43(33.9)	2(1.6)	
No	81(63.8)	116(90.6)	
Missing data	3(2.4)	10(7.8)	
DBP ≥ 90 mmHg postpartum (within 24 h)			<0.001
Yes	28(22.1)	2(1.6)	
No	96(75.6)	116(90.6)	
Missing data	3(2.4)	10(7.8)	
Occurrence of pulmonary oedema			
Yes	0(0)	0 (0)	
No	124 (97.6)	118 (92.2)	
Missing data	3(2.4)	10(7.8)	
Maternal death before hospital discharge post-delivery			
Yes	0(0)	0(0)	
No	124(97.6)	118 (92.2)	
Missing data	3(2.4)	10(7.8)	
Fetal outcome measures			
Fetal growth restriction			0.289
Yes	12(9.5)	7(5.5)	
No	109(85.8)	107(83.6)	
Missing data	6(4.7)	14(10.9)	
Gestational age (weeks) at delivery			0.396
Yes	38.0 ± 0.5	38.23 ± 0.4	
No			
Fetal compromise/ distress before delivery			0.300
Yes	19(15.0)	13(10.2)	
No	96(75.6)	98(76.6)	
Missing data	12(9.5)	17(13.3)	
Abruptio placenta			
Yes	0(0)	0 (0)	
No	112 (88.2)	112 (87.5)	
Missing data	15(11.8)	16(12.5)	
Apgar score in 1 min	8.1 ± 0.3	7.9 ± 0.218	0.255
Apgar score in 5 min	9.4 ± 0.3	9.2 ± 0.1	0.355
Birth weight (gram)	2986.6 ± 108.3	2982.8 ± 123.3	0.963
Admission to neonatal intensive care unit			0.231

Table 4 (*continued*)

Variable	Mean ± SD, or frequency (%)		p-value
	Stage 1 ACC/AHA hypertension (study arm) n = 127	Blood pressure less than stage 1 hypertension (control arm), n = 128	
Yes	7(5.5)	3(2.3)	
No	117(92.2)	114(89.1)	
Missing data	3(2.4)	11(8.6)	

Abbreviations: DBP, Diastolic blood pressure; SBP, Systolic blood pressure.

study has limitations. There were some missing data because of the retrospective design. The effects of these were reduced by having a sufficient sample size. Although the study and control arms were not statistically different in terms of comorbidities, future studies should exclude women with comorbidities that may affect the outcome measures. Notably, the following are some ways the comorbidities in the index study may influence the outcome measures. Anaemia, Human Immunodeficiency Virus infection, and previous cervical surgery such as large loop excision of transformation zone are plausible risk factors for preterm birth. Epileptic patients may undergo preterm birth, particularly those with poorly controlled seizures. A previous pregnancy loss, such as (miscarriage or intrauterine fetal demise) is a risk factor for a repeat pregnancy loss. A previous caesarean delivery may result in conditions such as intra-abdominal adhesions and placenta previa which predispose to adverse outcomes during childbirth. Additionally, suspected scar tenderness from a previous caesarean delivery may result in iatrogenic preterm birth. Previous pulmonary tuberculosis is a risk factor for chronic obstructive airway disease [15], which may be associated with fetal growth restriction and preterm birth. Finally, BMI above normal, diabetes, previous myocardial infarction, previous preeclampsia, and previous eclampsia are individual risk factors for HDP. Concerning these comorbidities, we are unable to provide detailed information about them, given the design of our study.

4.3. Interpretation

Progression to stage 2 hypertension (HDP) was more common among women with stage 1 hypertension (study arm) than those with lower BP (control arm), i.e., 47.2 % vs 1.6 %. This progression may account for the mean of the highest BPs during the prenatal period being highest among women with stage 1 hypertension (Table 3). Our findings are similar to other studies, which showed that stage 1 hypertension at less than 20 weeks' gestation is a risk factor for developing HDP [9,16,17]. For instance, Wu and colleagues in one of the largest studies on this topic (n = 47 874), found that 40.1 % of women with stage 1 hypertension (at less than 20 weeks' gestation) developed HDP while only 7.5 % of women with BP less than the threshold for stage 1 hypertension progressed to the same condition [9]. In another study, Darwin et al. (2021) reported that women identified to have stage 1 hypertension in the first trimester had higher rates of preeclampsia than those who were normotensive (11.4 % vs 3 %) [18]. Sutton et al., found that stage 1 hypertensive compared to normotensive women at 13–25 weeks' gestation had a statistically significant increased risk of resulting in preeclampsia (15 % vs 5.4 %), gestational diabetes (6.1 % vs 2.5 %) and preterm birth (4.2 % vs 1.1 %) [16]. Additionally, the risk of preeclampsia increases step-wise with an increasing category of BP before 20 weeks' gestation [18,19]. Notably, the findings of a few studies differ from ours and show that stage 1 hypertension before 20 weeks' gestation did not progress to HDP [20]. In our study, however, the commonest types of HDP that the women with stage 1 hypertension developed were gestational hypertension (29.1 %), preeclampsia (13.4 %), and transient gestational hypertension (4.7 %). González-Valencia et al., also reported that the commonest type of HDP that women with prehypertension before 20 weeks' gestation in their study suffered was gestational

hypertension (23.94 %), followed by preeclampsia (18.3 %) [21]. Other authors have also reported a progression to preeclampsia and gestational hypertension among women with stage 1 hypertension [22].

Again, the difference in the percentages of women with stage 1 hypertension that progressed to HDP (in various studies) may be due to the baseline risk of HDP. Most of our patients are of African ancestry, which is a risk factor for HDP. SA has a high burden of HDP [12,23], and it may not be surprising that approximately 47 % of the women progressed to develop HDP. This finding supports the recognition of prehypertension, also called stage 1b hypertension (systolic BP 135–139 mmHg and or diastolic BP 85–89 mmHg), as a risk factor for HDP in SA [5].

The findings from the present study also showed that more women in the study arm compared to those in the control arm developed BP above or equal to stage 2 hypertension within 24 h postpartum (Table 4): 43/127(33.9 %) vs 2/128(1.6 %). This was associated with an increase in the use of antihypertensives drug within 24 h postpartum, where at least 35/127(27.6 %) of the women in the study arm and 1/128(0.8 %) of those in the control arm received postpartum antihypertensive drug therapy. In a study by Black et al., (n = 5960), 1.1 % of participants with stage 1 hypertension developed postpartum hypertension [5]. Black et al., highlighted the importance of early postpartum screening and improved follow-up of the women as these measures may help prevent future cardiovascular diseases [5]. They noted the importance of more prospective studies, given that little is known about the long-term outcomes of these women that had postpartum hypertension. Coincidentally, the occurrence of 24.4 % of de novo postpartum hypertension has been reported among normotensive women (BP < 140/90 mmHg) in SA, and the authors detailed the appropriate management strategy for the condition [24]. However, the index study shows that stage 1 hypertension is a risk factor for HDP during both prenatal and postnatal periods. It is associated with increased hospitalization during pregnancy [21] and adverse pregnancy outcomes [25,26]. Therefore, women with this risk factor (stage I hypertension) require close monitoring during prenatal and postnatal periods. In support of this opinion, a study in Australia showed that stage 1 hypertension was among the cardiometabolic risk factors associated with increased risk of metabolic syndrome (aOR 5.5, 95 % CI 1.8 – 17.3, p = 0.004) and cardiovascular disease, ten years postpartum [27]. Unfortunately, the long-term clinical course of stage 1 hypertension in our cohort is unknown. We recommend future multicentre studies that will follow-up women with stage 1 hypertension for a more extended period postpartum.

The fetal outcomes did not show any statistically significant difference between the two groups. This is similar to other studies which showed that fetal outcomes at birth were not affected by stage 1 hypertension [8]. In contrast, other authors have demonstrated an increased risk of fetal affectation if stage 1 hypertension occurs earlier in pregnancy than later [25,28]. For instance, stage 1 hypertension in early pregnancy has been associated with an increased risk of small for gestational age and preterm birth [22]. However, the association between fetal affectation and early-onset stage 1 hypertension was not demonstrated in another study which showed that 59 % of women diagnosed with stage 1 hypertension particularly in late pregnancy had babies that were small for gestational age [29]. Nonetheless, a *meta*-analysis published in 2021 showed that stage 1 hypertension increases the risk of small for gestational age, preterm birth, early-term birth, and low birth weight [30]. Notably, other authors have reported that the risk of severe maternal and neonatal morbidity is increased only by stage 2 and not stage 1 hypertension [19]. In the authors' opinion, a longitudinal study with a long observation period is required to completely study the possible affectation of the babies born to mothers who had stage 1 hypertension during pregnancy.

5. Conclusion

The occurrence of stage 1 hypertension in the first 20 weeks of pregnancy is a risk factor for the development of stage 2 hypertension in the antenatal and postnatal periods; and increases the use of antihypertensive drug therapy. It has the potential to worsen adverse maternal outcomes. Therefore, women with stage 1 hypertension require close monitoring during pregnancy and postpartum periods.

Contribution of authors

MRM and NCN conceived the study. MRM collected data. The initial manuscript was drafted by MRM, and all authors revised and approved the final version submitted. The study was supervised by NCN (the main supervisor) and LC (the co-supervisor).

Declaration

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Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.preghy.2023.12.002.

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